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New Pharmacological Strategies in some Heart Disease Under a Toxicological Point of View

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Introduction

Today we need to introduce new approach in some cardiovascular pathology and to do this we think is useful to use a toxicological approach, new delivery systems and new diagnostic strategies. (This paper is produced under a pharmaceutical-toxicological approach.)

Material and Methods

With a review method observing some literature in biomedical database we try to produce new approach in pharmacological strategies in some relevant heart disease or to prevent it.

Results

From literature we have finded

We can think a new method in heart disease clinical staging strategy. New tests that can make possible to stress cardiac metabolism in normal, lowland high working conditions or in para-phisio-pathological conditions but in local place (heart situation and not in plasma in example). We think that this new methods can be useful in much heart disease and condition to prevent some events. (Heart attacks, ischemic disease, aritmia, heart failure, transplants, sports and so on). Every bio-medical discipline has specific diagnostic discipline, but it can be useful to translate the various diagnostic system strategies from a discipline to other: this make possible to observe phenomena with a different point of view [1].

"We must consider an endogenous local toxicology aspect time related to verify some pathologic process and phenomena under a new useful light. In some time related CV local metabolic-catabolic-toxic status we can observe some cellular effect resulting in global organ- apparatus failure. The time involved (and kinetics aspect) in resolve some temporary metabolic-catabolic- electric gradients or the global velocity involved in this process can be fundamental to consider (too rapid evolution or too slow reduction in balancing -equilibrates some physiologic systems)" [2].

In example what happen in SCD in untrained? [1] Why physical training can reduce this event (SCD)? We can think a condition of ENDOGENUS toxicity time- KINETICKS related. So We think in this kind of condition is relevant to better observe the time related endogenous intra- toxicity situation involved in some Metabolic- catabolic –electrical cell membrane and other.

For example involved in some heart aritmia, epilectic status, septic shock and other situation high time related (ischemic coronaric spasm etc). In embryology, oncology, toxicology, pathology some heart and brain disease the time is relevant factors added to endogenous local- micro environment and inter-cellular communication (crisis). We can consider an intra- local toxicology aspect time related to better verify some pathologic process under a new light [3].

In actual pharmacological therapy we can see that some drugs can be added to other medical instruments to improve their activity: in example we can see medicated stent for some coronary disease, or hormonal medical devices used in pregnancy prevention, but other example is known today. In example Carmustine wafer is delivered by delivery systems in some brain cancer and radioactive seed implants in prostatic cancer. Ocular intra virtual implants for some macular degeneration (MABS or

cortisones) other implant delivery systems drugs, naltrexone implant for opiate dependence.

Other strategies imply carrier use to deliver the drugs in the site of action

In example MABS linked to radioactive isotopes in some relapse of severe Hodgkin disease but many other examples we can see in therapy used today. So we can think that other chronic conditions can be treated using a combination of drugs with other instrument to improve the clinical outcomes. This is to make possible that the ERLICH MUGIC BULLETS can act in the right site reducing the side effect. In example today we can see various medical interventional radiological strategy to treat in coronary and heart disease with medicate stents positioning or to local use of contrast agents or other valve surgery procedures with global good clinical results [4].

"We can say that under the light of the article finded in this paper but also to other works published we can think a new system "to regenerate" a valve tissue calcified. We think that adding 3 strategies medical interventional radiological strategy, pharmacological agent like complexant or other molecule with a delivery system we can have relevant effect reducing global toxicology" [4]

"The causes and the mechanisms underlying the development of CAS are still poorly defined and are likely multifactorial. In the 1980s, the autonomic nervous system was found to play an important role in the pathophysiology of CAS. In the 1990 YEARS, some factors as flogosis inflammation, endothelial vessel dysfunction, oxidative stress agents, respiratory alkalosis and magnesium (Mg) deficiency were identified as predisposing factors In the late 1990s and early 2000s, genetic mutations were found to be associated with CAS. Nonetheless, coronary vascular smooth muscle cell hyper-reactivity seems to constitute the substrate for CAS" [6].

"Sudden death is an important but widely under-recognized consequence of stroke. Acute stroke can disturb central autonomic control, resulting in myocardial injury, electrocardiographic abnormalities, cardiac arrhythmias, and ultimately sudden death. Experimental - clinical objective evidence suggests that autonomic imbalance situations is more frequent after brain infarcts involving the insular cortex region, crucial for the control of autonomic functions level (sympathetic – parasympathetic). Cardiovascular comorbidities increase the risk of cardiac morbidity and mortality after stroke [7]. Thus, many sudden deaths and serious non-fatal cardiac events after stroke are probably due to an interaction between cardiovascular and neurological causes. The exact mechanisms leading to sudden death remain incompletely understood. Further research is needed to investigate the autonomic consequences of acute stroke and to identify patients at high risk of sudden death" [8].

Results of the first large-scale randomized trial of this treatment. "TACT a large-scale clinical trial of chelation therapy for some atherosclerotic coronary pathology, found that EDTA (a chelation therapy) reduced the risk of a composite of adverse CV clinical outcomes, especially among patients with diabetes. Before disodium EDTA chelation can take its place among other accepted therapies in the routine care of post-MI patients, however, it is important that further replicative and mechanistic clinical trials be performed" [9].

Evidence that human cardiac dysfunction is associated with excess lipid; Clinical data show that both obesity and diabetes markedly increase risk of heart failure even in the absence of ischemic vascular disease. The molecular mechanisms could be either increased the lipid uptake or an impaired mitochondrial oxidative function leading to accumulation of molecules of TGs and toxic lipid species such as ceramides, which cause myocyte loss through apoptosis, induction of iNOS and pro-hypertrophic signaling. Therefore, the specific form of excess cardiac lipid products- compounds their cellular compartmentalization and storage form (lipid droplets), and the specific cause of CHF heart failure are likely to determine the importance of lipotoxicity in human disease. Studies of human HAERT function / metabolism rely on imaging methods (relatively non-invasive). PET scanning DIAGNOSTIC assesses the uptake of various tracers into the heart (well standardized for the glucose and FFA uptake).

Myocardial PET imaging technique has consistently showed the increased FFA uptake and oxidation, impaired glucose uptake, in diabetic patients with normal systolic and mildly impaired diastolic function.

In Recent time RM magnetic resonance protocols have been developed to track TG metabolism such as 1H magnetic resonance spectroscopy (MRS). Although hypertension and coronary artery disease are common in obese and diabetic patients, reduced heart function independent of these underlying disorders may relate to toxicities from excess metabolic substrates and defective insulin action. Some studies in patients with obesity and diabetes correlated TG accumulation with left ventricular hypertrophy [10]. More TG has also been found in failing hearts of patients with obesity or diabetes at the time of transplantation [11]. Reducing plasma lipids to reduce lipid uptake and converting oxidation to more glucose and less FA might be a method to treat patients with lipotoxic and ischemic heart failure. Agents that inhibit FA oxidation have been used for angina [12].

CV diseases are a leading cause of morbidity and mortality in most developed countries of the world.

DRUGS illicit drugs and toxins can significantly contribute to the overall cardiovascular burden. The compounds are in this paper classified into agents that have significant effects on the heart, blood vessels, or both. The mechanism(s) of toxic action are discussed and the treatment modalities are briefly mentioned in relevant cases.

Due to the large number of clinically relevant compounds and molecules discussed, this paper could be of interest to a broad audience including pharmacologists toxicologists, clinical

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pharmacists, physicians, and medicinal chemists, medical laboratory professionals and other particular emphasis is given to the clinically relevant topics and interesting including the cardiovascular toxicity of illicit sympathomimetic drugs (e.g., cocaine, amphetamines, cathinones), drugs that prolong the QT interval, antidysrhythmic drugs, digoxin and others molecules, cardio-active steroids, beta blockers, CA++ channel blockers, female hormones, FANS nonsteroidal anti-inflammatory, and anticancer compounds as anthracyclines and targeted therapy interfering with the HER2 or the vascular endothelial growth factor pathway [13]. Diabetes and heart failure PATHOLOGY commonly coexist and portend worsened prognosis than either disease alone [14].

Discussion & Conclusion

Observing the reported literature in some heart disease we have see that some phenomena are deeply involved: Kinetics and gradients in metabolism catabolism time related toxic like effect electrical cell membrane status, smooth vascular muscle cell hyper-reactivity platelet iperactivations, central autonomic control after acute stroke great electrolytes unbalances et other. We think that using and antidotes- toxicological approach new delivery systems and new diagnostic approach in ranking the clinical risk we can obtain new Pharmacological strategies useful in some cardiovascular conditions. To prevent some kind of heart disease we think can be useful introduce new diagnostic strategy to verify in stressing conditions the local metabolic heart performance in example in young or before high sports activities. Can we consider a sort of chronic endogenous poisoning some cardiovascular disease as atherosclerotic pathology or diabetes type II or other?

We think that a deep knowledge in the right mechanism(s) of toxic action in some cardio vascular conditions using a specific toxicological approach can produce new research hypothesis to be verified for new pharmacological way to be introduced.

Clarification

This paper has not any diagnostics or therapy intent only to produce new research hypothesis.

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